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SPINAL CORD INJURY: A UNCOMMON CAUSE OF ACQUIRED PREMATURE EJACULATION

Hypothesis / aims of study

Acquired premature ejaculation (PE) has been recently defined as “a clinically significant reduction in latency time, often to about 3 minutes or less and the inability to delay ejaculation on all or nearly all vaginal penetrations, and responsible for negative personal consequences, such as distress, bother, frustration and/or the avoidance of sexual intimacy” (1). Anejaculation during masturbation or coïtus occurs in 88% and 67% of men with complete and incomplete spinal cord injury (SCI) respectively (2). Conversely, very seldom SCI men complain of PE. The aim of this study was to report the first comprehensive series of post-SCI acquired PE.

Study design, materials and methods

We have conducted a retrospective study based on the review of medical records of patients reporting PE followed in two physical medicine and rehabilitation centers from 1986 to 2014.

The following clinical data have been collected in SCI patients: age at SCI, time elapsed since SCI, etiology of the SCI, ASIA impairment scale (AIS) specifying the completeness and upper limit of the spinal lesion and Urodynamic data. In SCI patients complaining about PE a structured interview by a trained physician regarding sexual function has been conducted.

Sexual dysfunction or absence of sexual experience prior to SCI, history of surgical intervention for spastic hypertonia, neuropathic bladder or neuropathic pain were exclusion criteria.

Primary endpoint was to describe clinical and urodynamic parameters associated to PE in SCI patients. The existence of a specific pattern of spinal lesion has been searched and correlations between i) the lesion’s characteristics (completeness, traumatic injury, lesion of thoraco-lumbar sympathetic and/or lumbar spinal generator of ejaculation and/or sacral parasympathetic and/or somatic centers) and ii) characterization of ejaculation (intravaginal premature ejaculation (IVPE) or anteportal ejaculation (AE), dribbling or forceful ejaculation, ability to control ejaculation and orgasm have been assessed.

Statistical analyses were performed with nonparametric tests: Fisher’s exact test to compare proportions, Pearson correlation test to assess correlation between variables. Significance was set at a level of less than 0.05.

Results

Forty-seven out of 2530 SCI patients have reported post-SCI acquired PE. Two patients were excluded because PE occurred following a dorsal root entry zone lesions for the treatment of neurogenic pain. Mean age at SCI was 33.3 +/- 13.3 years. 14 patients (31%) visited the out-patient clinic specifically because of negative personal consequences of PE, including infertility in 4. In 31, PE has been diagnosed during initial assessment or follow up of neurogenic detrusor overactivity (NDO).

Characteristics of SCI (figure1)

SCI always involved the lower spinal cord with an upper limit below T9. SCI was complete in 18 patients (40 %) and incomplete in 27 (60 %). Completeness was positively correlated with traumatic SCI (r=-0.39, p=0.008) (table 1).

Description of PE

Ejaculation was never spontaneous but always triggered by psychogenic and/or genital stimulation. The median duration between SCI and the first PE episode was 3.5 +/- 35.5 months (0.2 to 216). Forty-two patients (93%) reported AE and 14 of them (33%) regained intravaginal ejaculation overtime, without treatment. For 3 patients (7%) ejaculation was intravaginal (IVPE) at the very beginning after SCI with a mean IELT of 1.4 min (0.2 to 3). The ability to control ejaculation was negatively correlated with traumatic SCI (r=-0.41, p=0.05). PE, either IVPE or AE, occurred since the very first sexual activity following SCI in all but one patient, who had a conus medularis ependymoma. Ejaculation, either IVPE or AE, was described as normal by only 1 patient, with decreased forcefulness by 5 patients (11%) and dribbling by 39 (87%). There was a positive correlation between the forcefulness of ejaculation and a lesser degree of completeness of SCI (r=0.33; p=0.03). All patients with a complete SCI reported dribbling ejaculations. Nine patients (20%) reported dribbling ejaculation without any pleasurable feeling, triggered by erotic thoughts, without any sexual contact. These patients had a different lesion pattern when compared to patients with ejaculation occurring in response to sexual contact: i) complete SCI in 7 (78%) vs 11 (31%, p=0.02), ii) complete or incomplete lesion of T12-L2 segments in all vs 2 (22%, p<0.001), iii) complete lesion of L3, L4 and/or L5 segments in 7 (78%) vs 4 (11%, p<0.001), iv) complete lesion of the S2-S4 segments in 5 (56%) vs 1 (3%, p<0.001).

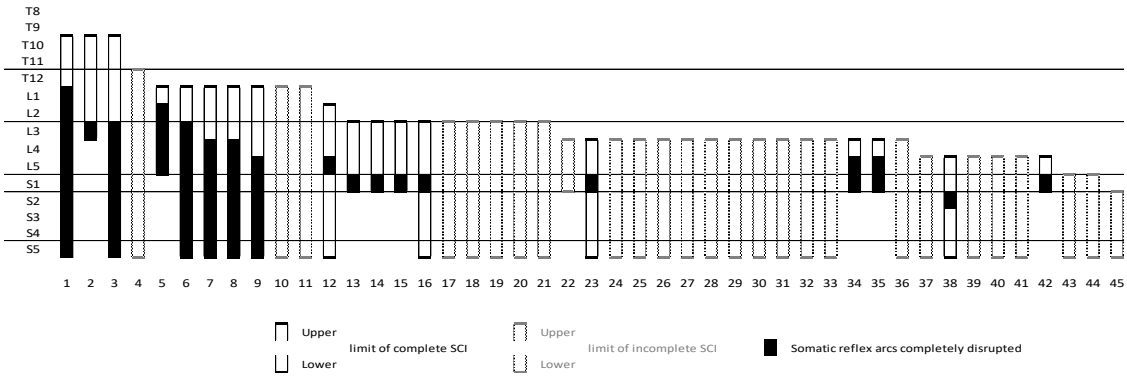

Figure 1. Location, extent and completeness of spinal cord lesion in 45 post-SCI acquired PE patients. X axis: patients, Y axis: spinal segments

Interpretation of results

Acquired PE in SCI patients remains a very rare condition, with only very few reports in literature. Despite that SCI is usually responsible for anejaculation this series provides robust evidences that acquired PE, most often anteportal, can be a consequence of SCI. Indeed, in our cohort, none of the patients reported PE prior to SCI.

The understanding of the neurophysiology of ejaculation has greatly evolved since the discovery of a spinal generator or ejaculation (SGE) ref. SGE, described is located in the L3-L5 segments (3). SGE coordinates the activation of spinal parasympathetic (S2-S4 segments) sympathetic (T12-L2 segments) and somatic ejaculation centers (S2-S4 segments).

Common features for the intraspinal lesions of PE SCI men are: (i) all patients had at least incomplete sparing of T12-L2 segments explaining preservation of semen secretion and seminal tract contractions involved in emission. (ii) L3-L5 segments were lesioned in almost all SCI men with PE. Nevertheless, these segments were never completely lesioned in patients with IVPE. We thus propose that severe SGE lesion impairs the mandatory coordination for normal ejaculation between the spinal autonomic and somatic centers. We postulate that these lesions are responsible for an imbalance between inhibitory and/or excitatory influences onto the SGE from the periphery (ascending inputs) and/or the brain (descending inputs), with a potential decrease in inhibitory inputs from the genitalia, conveyed by C fibers post-SCI reorganization leading to PE. Indeed, in our cohort, NDO occurred in 78% of the patients, after a delay similar to PE occurrence. (iii) Sacral segments were lesioned but with an unusual pattern. Indeed, parasympathetic reflex arc remained functional, as evidenced by NDO in 78% of patients, conversely, S2-S4 somatic reflex arcs were partially or completely disrupted in 80% of patients leading to paralysis of the striated pelvi-perineal musculature leading to dribbling ejaculation.

Concluding message

PE consecutive to SCI is a very rare and often severe condition with AE being common. It likely involves an imbalance between activator and inhibitor inputs onto the spinal centers of ejaculation with a central role for the SGE. While uncommon, SCI is an etiology of acquired PE.

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Disclosures

Funding: Nothing to declare **Clinical Trial:** Yes **Public Registry:** No **RCT:** No **Subjects:** HUMAN **Ethics not Req'd:** Retrospective study No intervention **Helsinki:** Yes **Informed Consent:** Yes